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Set	Items	Description
S1	140254	PARTICULATE?? OR (ALUMINUM(W)HYDROXIDE) OR (COLLOIDAL(W)GO-LD) OR (POLYSTYRENE(W)LATEX)
S2	296739	AGGREGAT?
S3	3984	S1 AND S2
S4	288154	SUGAR OR TREHALOSE OR CARBOHYDRATE
S5	75	S3 AND S4
S6	57	RD (unique items)
S7	25	S6 AND PY<=1994
S8	4715057	PREVENT? OR REDUC?
S9	7	S7 AND S8
? s scavenger(5n)radical		
	30016	SCAVENGER
	359852	RADICAL
S10	9049	SCAVENGER(5N)RADICAL
? s s4 and s10		
	288154	S4
	9049	S10
S11	100	S4 AND S10
? s mannitol		
S12	30088	MANNITOL
? s s12 and s10		
	30088	S12
	9049	S10
S13	558	S12 AND S10
? s s13 and py<=1994		
Processing		
	558	S13
	25468139	PY<=1994
S14	201	S13 AND PY<=1994
? s s14 and s1		
	201	S14
	140254	S1
S15	1	S14 AND S1
? t s15/3,k,ab/1		

15/3,K,AB/1 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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03315480 Genuine Article#: NW488 Number of References: 44
Title: EFFECT OF SCAVENGERS OF ACTIVE OXYGEN SPECIES ON CELL-DAMAGE CAUSED
IN CHO-K1 CELLS BY PHENYLHYDROQUINONE, AN O-PHENYLPHENOL METABOLITE (Abstract Available)
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Abstract: Phenylhydroquinone (PHQ), a metabolite of o-phenylphenol (OPP), is easily autooxidized to phenylbenzoquinone (PBQ) via the semiquinone (phenylsemiquinone, PSQ) with concomitant production of superoxide anion radicals (O-2(radical-anion)) We have used scavengers of active oxygen species to examine whether or not O-2(radical-anion) produced during oxidation of PHQ is related to cell damage in CHO-K1 cells. PHQ at 10 μ g/ml (3-h treatment) induced sister-chromatid exchange (SCE), endoreduplication (ERD) and cell-cycle delay in CHO-K1 cells. These effects were inhibited by catalase (280 U/ml), a scavenger of hydrogen peroxide (H2O2), as well as by the reductants, ascorbate (3 mM) and GSH (1 mM). Mannitol (50 mM), a scavenger of hydroxyl radical (OH.), was ineffective and superoxide dismutase (SOD, 150

U/ml), a **scavenger** of O-2(**radical-anion**), or SOD plus catalase rather intensified the toxicity as did aminotriazole (20 mM), an inhibitor of catalase. Analyses of incubation solutions by HPLC showed that the extent of cell damage is correlated with PHQ loss; catalase suppressed PHQ loss, whereas SOD promoted it. The correlation was more clearly seen in the time courses of cell death and PHQ loss during incubation of PHQ with each of the scavengers of active oxygen species. These results show that neither O-2(**radical-anion**) nor OH. participates in the cell damage, but rather H2O2 generated via dismutation of O-2(**radical-anion**) may participate, probably by accelerating the autoxidation of PHQ and thus causing an increase in the production of toxic intermediates. In fact, conversion of PHQ to PBQ, a reactive product, was demonstrated during incubation with PHQ in phosphate-buffered saline by following the changes in W-visible spectra of PHQ. Inclusion of H2O2 (0.2 or 1 mM) in the incubation mixture accelerated the PHQ loss. The present results can be explained in terms of the autoxidation mechanism of hydroquinone proposed by O'Brien (1991). Different from the results in the absence of S9 mix, the cell damage induced by 50 µg/ml OPP in the presence of S9 mix was not influenced by any of the scavengers of active oxygen species used. We conclude that PHQ causes cytotoxic and genotoxic effects through its autoxidation, both enzymatic and nonenzymatic, and that reactive intermediate(s) such as PSQ and/or PBQ may be ultimately responsible for the effects. H2O2 formed during the oxidation process participates in the damaging effects caused in the absence of S9 mix, probably by accelerating the autoxidation.

, 1994

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Research Fronts: 92-0790 001 (BIOASSAY-DIRECTED CHEMICAL-ANALYSIS OF AMBIENT AIR **PARTICULATE** EXTRACTS; SALMONELLA MUTAGENICITY; ATMOSPHERIC POLYCYCLIC AROMATIC-HYDROCARBONS; AMES TEST)

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